Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	8033	(435/7.1,7.92-7.95,420).CCLS.	USPAT; EPO	OR	OFF	2005/01/03 17:24
L2	4362	PTH or (parathyroid adj1 homrone)	USPAT; EPO	OR	OFF	2005/01/03 17:24
L3	11	l2 same antibody same whole	USPAT; EPO	OR	OFF	2005/01/03 17:24
L4	11	l2 and l3	USPAT; EPO	OR	OFF	2005/01/03 17:24
L5	143	l1 and l2	USPAT; EPO	OR	OFF	2005/01/03 17:25
L6	95	l5 and whole	USPAT; EPO	OR	OFF	2005/01/03 17:25

```
Connecting via Winsock to STN



Welcome to STN International!  Enter x:x

LOGINID:ssspta1641cxc

PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2


* * * * * * * * * *   Welcome to STN International   * * * * * * * * * *

 NEWS  1           Web Page URLs for STN Seminar Schedule - N. America
 NEWS  2           "Ask CAS" for self-help around the clock
 NEWS  3  SEP 01   New pricing for the Save Answers for SciFinder Wizard within
                   STN Express with Discover!
 NEWS  4  OCT 28   KOREAPAT now available on STN
 NEWS  5  NOV 30   PHAR reloaded with additional data
 NEWS  6  DEC 01   LISA now available on STN
 NEWS  7  DEC 09   12 databases to be removed from STN on December 31, 2004
 NEWS  8  DEC 15   MEDLINE update schedule for December 2004
 NEWS  9  DEC 17   ELCOM reloaded; updating to resume; current-awareness
                   alerts (SDIs) affected
 NEWS  10 DEC 17   COMPUAB reloaded; updating to resume; current-awareness
                   alerts (SDIs) affected
 NEWS  11 DEC 17   SOLIDSTATE reloaded; updating to resume; current-awareness
                   alerts (SDIs) affected
 NEWS  12 DEC 17   CERAB reloaded; updating to resume; current-awareness
                   alerts (SDIs) affected
 NEWS  13 DEC 17   THREE NEW FIELDS ADDED TO IFIPAT/IFIUDB/IFICDB
 NEWS  14 DEC 30   EPFULL: New patent full text database to be available on STN
 NEWS  15 DEC 30   CAPLUS - PATENT COVERAGE EXPANDED

 NEWS EXPRESS  OCTOBER 29 CURRENT WINDOWS VERSION IS V7.01A, CURRENT
               MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
               AND CURRENT DISCOVER FILE IS DATED 11 AUGUST 2004
 NEWS HOURS    STN Operating Hours Plus Help Desk Availability
 NEWS INTER    General Internet Information
 NEWS LOGIN    Welcome Banner and News Items
 NEWS PHONE    Direct Dial and Telecommunication Network Access to STN
 NEWS WWW      CAS World Wide Web Site (general information)

Enter NEWS followed by the item number or name to see news on that
specific topic.

  All use of STN is subject to the provisions of the STN Customer
  agreement.  Please note that this agreement limits use to scientific
  research.  Use for software development or design or implementation
  of commercial gateways or other similar uses is prohibited and may
  result in loss of user privileges and other penalties.

  * * * * * * * * * * * * * * STN  Columbus  * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 16:05:37 ON 03 JAN 2005

=> file .meeting
'EVENTLINE' IS NOT A VALID FILE NAME
Enter "HELP FILE NAMES" at an arrow prompt (=>) for a list of files
that are available.  If you have requested multiple files, you can
specify a corrected file name or you can enter "IGNORE" to continue
```

```
accessing the remaining file names entered.
ENTER A FILE NAME OR (IGNORE):ignore
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                        ENTRY    SESSION
FULL ESTIMATED COST                                      0.21       0.21

FILE 'AGRICOLA' ENTERED AT 16:05:52 ON 03 JAN 2005

FILE 'BIOTECHNO' ENTERED AT 16:05:52 ON 03 JAN 2005
COPYRIGHT (C) 2005 Elsevier Science B.V., Amsterdam. All rights reserved.

FILE 'CONFSCI' ENTERED AT 16:05:52 ON 03 JAN 2005
COPYRIGHT (C) 2005 Cambridge Scientific Abstracts (CSA)

FILE 'HEALSAFE' ENTERED AT 16:05:52 ON 03 JAN 2005
COPYRIGHT (C) 2005 Cambridge Scientific Abstracts (CSA)


FILE 'IMSDRUGCONF' ENTERED AT 16:05:52 ON 03 JAN 2005
COPYRIGHT (C) 2005 IMSWORLD Publications Ltd.

FILE 'LIFESCI' ENTERED AT 16:05:52 ON 03 JAN 2005
COPYRIGHT (C) 2005 Cambridge Scientific Abstracts (CSA)


FILE 'MEDICONF' ENTERED AT 16:05:52 ON 03 JAN 2005
COPYRIGHT (c) 2005 FAIRBASE Datenbank GmbH, Hannover, Germany

FILE 'PASCAL' ENTERED AT 16:05:52 ON 03 JAN 2005
Any reproduction or dissemination in part or in full,
by means of any process and on any support whatsoever
is prohibited without the prior written agreement of INIST-CNRS.
COPYRIGHT (C) 2005 INIST-CNRS. All rights reserved.

=> antibody and (pth or parathyroid hormone) and whole and non-whole
L1             0 FILE AGRICOLA
L2             0 FILE BIOTECHNO
L3             0 FILE CONFSCI
L4             0 FILE HEALSAFE
L5             0 FILE IMSDRUGCONF
L6             0 FILE LIFESCI
L7             0 FILE MEDICONF
L8             0 FILE PASCAL

TOTAL FOR ALL FILES
L9             0 ANTIBODY AND (PTH OR PARATHYROID HORMONE) AND WHOLE AND NON-WHOL
                 E

=> antibody and (pth or parathyroid hormone) and whole
L10            0 FILE AGRICOLA
L11            9 FILE BIOTECHNO
L12            0 FILE CONFSCI
L13            0 FILE HEALSAFE
L14            0 FILE IMSDRUGCONF
L15            3 FILE LIFESCI
L16            0 FILE MEDICONF
L17            7 FILE PASCAL

TOTAL FOR ALL FILES
L18           19 ANTIBODY AND (PTH OR PARATHYROID HORMONE) AND WHOLE

=> dup rem
ENTER L# LIST OR (END):l19
L19 IS NOT VALID HERE
```

The L-number entered has not been defined in this session, or it has been deleted. To see the L-numbers currently defined in this session, enter DISPLAY HISTORY at an arrow prompt (=>).

=> dup rem
ENTER L# LIST OR (END):l18
DUPLICATE IS NOT AVAILABLE IN 'IMSDRUGCONF, MEDICONF'.
ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE
PROCESSING COMPLETED FOR L18
L19 11 DUP REM L18 (8 DUPLICATES REMOVED)

=> d l19 ibib abs total

L19 ANSWER 1 OF 11 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
DUPLICATE
ACCESSION NUMBER: 2003:36432259 BIOTECHNO
TITLE: Influence of **PTH** assay methodology on differential diagnosis of renal bone disease
AUTHOR: Reichel H.; Esser A.; Roth H.-J.; Schmidt-Gayk H.
CORPORATE SOURCE: Dr. H. Reichel, Nephrological Center, Schramberger Strasse 28, D-78054 Villingen-Schwenningen, Germany. E-mail: helmut.reichel@dialyse-schwenningen.de
SOURCE: Nephrology Dialysis Transplantation, (01 APR 2003), 18/4 (759-768), 20 reference(s)
CODEN: NDTREA ISSN: 0931-0509
DOCUMENT TYPE: Journal; Article
COUNTRY: United Kingdom
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2003:36432259 BIOTECHNO
AB Background. Determination of plasma **parathyroid hormone** (**PTH**) is routinely performed to diagnose and monitor renal bone disease. Recently, a new **PTH** assay ('**whole PTH**') using an **antibody** directed specifically against **PTH**(1-4) has been introduced. It was the aim of the current study to evaluate **whole PTH** and parameters derived from **whole PTH** in renal bone disease. Methods. The following measurements were carried out in blood samples from 141 unselected haemodialysis patients: three intact **PTH** assays (Nichols, Roche Elecsys®, Scantibodies total); **whole PTH** (Scantibodies); bone-specific alkaline phosphatase (bAP); tartrate-resistant acid-phosphatase 5b (TRAP 5b); osteocalcin, 25-hydroxyvitamin D. Parameters derived from **whole PTH** were: (i) non-**PTH**(1-84), difference between intact **PTH** (Scantibodies assay) and **whole PTH**; (ii) **whole PTH**/non-**PTH**(1-84) ratio. Results. The values generated by the intact **PTH** assays were comparable. The mean **whole PTH** concentration was lower than mean intact **PTH** concentrations (16.9 ± 18.1 vs 26.4 ± 30.5 pmol/l, Nichols, $P < 0.05$). The correlation coefficients between all four **PTH** assays were comparable and were very high ($r > 0.96$, ns). The rank order of values generated by the **whole PTH** assay was statistically not significantly different from the rank order generated by the Nichols intact **PTH** assay. The median non-**PTH**(1-84) concentration was 5.2 pmol/l (range 0-49.4). All **PTH** assays correlated highly significantly with non-**PTH** (1-84) (correlation coefficients 0.83-0.92). Corrected serum calcium was also associated with non-**PTH** (1-84) but the correlation was weaker ($r = 0.28$). Regression analysis indicated that the non-**PTH** (1-84) concentration could be predicted by 76.6-84.6% by the prevailing intact **PTH** concentrations. Other parameters contributed only marginally to prediction of non-**PTH**(1-84). In the entire patient group, there was no statistically significant correlation between the **whole PTH**/non-**PTH**(1-84) ratio and any

of the **PTH** assays or biochemical bone markers. Eight of 141 patients had a **whole PTH**/non-**PTH**(1-84) ratio < 1. TRAP 5b, bAP and osteocalcin had high correlations with intact **PTH** assays and the **whole PTH** assay (correlation coefficients 0.51-0.56, no significant difference). None of the **PTH** assays was superior to any other **PTH** assay in predicting serum concentrations of the bone markers. Therapy with active vitamin D metabolites (n = 70) did not alter the results of our analyses. Conclusions. With respect to information about bone turnover we were not able to find differences between **whole PTH** and intact **PTH** assays. Our data also suggest that **whole PTH** and intact **PTH** assays give similar information. (i) The correlation between all **PTH** assays was very high. (ii) The rank order between **whole PTH** and Nichols intact **PTH** assays was comparable. (iii) The association between intact **PTH** assays and non-**PTH**(1-84) was very high. Albeit non-**PTH**(1-84) was mostly determined by the prevailing intact **PTH** concentration, diagnostic information on parathyroid activity provided by **whole PTH** or intact **PTH**, respectively, may differ in individual patients. How often this would happen cannot be answered with the currently available data. Unequivocal structural identification of the non-**PTH**(1-84) fraction would facilitate the answer to that question. The use of the **whole PTH**/non-**PTH**(1-84) ratio as a biochemical bone marker in renal bone disease requires further investigation.

L19 ANSWER 2 OF 11 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
DUPLICATE

ACCESSION NUMBER: 2003:36592205 BIOTECHNO
TITLE: **Parathyroid hormone** assays - Evolution and revolutions in the care of dialysis patients
AUTHOR: Malluche H.H.; Mawad H.; Trueba D.; Monier-Faugere M.-C.
CORPORATE SOURCE: Dr. H.H. Malluche, Div Nephrology Bone/Mineral Metab., UK Medical Center, University of Kentucky, 800 Rose Street, Lexington, KY 40536-0084, United States. E-mail: hhmall@pop.uky.edu
SOURCE: Clinical Nephrology, (01 MAY 2003), 59/5 (313-318), 45 reference(s)
CODEN: CLNHBI ISSN: 0301-0430
DOCUMENT TYPE: Journal; General Review
COUNTRY: Germany, Federal Republic of
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2003:36592205 BIOTECHNO
AB Renal osteodystrophy may present with low, normal, or high bone turnover. An ideal **parathyroid hormone** (**PTH**) assay should discriminate between the bioactive **whole PTH** -(1-84) molecule and **PTH** fragments, including the **PTH** -(7-84) fragment. Most dialysis patients have "intact" **PTH** (iPTH) levels between 65 and 450 pg/ml, which are poorly predictive of bone turnover state, making the iPTH test of limited value for bone turnover prediction. iPTH levels higher than 500 pg/ml can be observed in some dialysis patients with low bone turnover, while iPTH levels as low as 100 pg/ml have been found in patients with bone turnover above normal, indicating the standard second generation iPTH assay is not a reliable sole indicator of bone turnover. The **whole PTH** immunoradiometric assay, a third generation assay, uses a detection **antibody** that recognizes antigenic determinants at the extreme amino-terminal (1-4) end of the **PTH** molecule, making the assay specific for biologically active **whole PTH**-(1-84). Comparing results using the **whole PTH** and iPTH assays, the **PTH**-(7-84) level is indirectly determined and the

PTH-(1-84)/**PTH**-(7-84) ratio can be calculated. It was shown that **PTH**-(7-84) inhibits the calcemic effect of **PTH**-(1-84) and its stimulatory effect on bone turnover. In the interpretation of results using the **PTH**-(1-84)/**PTH**-(7-84) ratio, it must be taken into consideration that second generation "intact" **PTH** assays have different cross-reactivity with **PTH**-(7-84). Therefore, when comparing or analyzing **PTH**-(1-84)/**PTH**-(7-84) ratios, the employed **PTH** assays must be identical. The **whole PTH** assay and the **PTH**-(1-84)/**PTH**-(7-84) ratio allow more meaningful interpretation of **PTH** trends, and offer a noninvasive means to more accurately diagnose bone disease in this population.

L19 ANSWER 3 OF 11 PASCAL COPYRIGHT 2005 INIST-CNRS. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 2003-0047687 PASCAL
COPYRIGHT NOTICE: Copyright .COPYRGT. 2003 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): Comparison of intact and '**whole** molecule' **parathyroid hormone** assays in patients with histologically confirmed post-renal transplant osteodystrophy
AUTHOR: GODBER Ian M.; PARKER Cornelle R.; LAWSON Nigel; HITCH Tony; PORTER Christine J.; ROE Simon D.; CASSIDY Michael J. D.; HOSKING David J.
CORPORATE SOURCE: Department of Clinical Chemistry, Nottingham City Hospital, Hucknall Road, Nottingham NG5 1PB, United Kingdom; Division of Mineral Metabolism, Nottingham City Hospital, Hucknall Road, Nottingham NG5 1PB, United Kingdom; Nottingham Renal Unit, Nottingham City Hospital Hucknall Road, Nottingham NG5 1PB, United Kingdom
SOURCE: Annals of clinical biochemistry, (2002), 39(p.3), 314-317, 8 refs.
ISSN: 0004-5632 CODEN: ACBOBU
DOCUMENT TYPE: Journal; Short communication
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United Kingdom
LANGUAGE: English
AVAILABILITY: INIST-17507, 354000104964540200
AN 2003-0047687 PASCAL
CP Copyright .COPYRGT. 2003 INIST-CNRS. All rights reserved.
AB Background Fragments of **parathyroid hormone** (**PTH**) have been identified (amino acids 7-84) which may interfere with commercially available 'intact molecule' **PTH** assays. Novel assays which employ an **antibody** directed to the first seven amino acids of the N-terminus of **PTH** are thought to be free from cross-reactivity with the 7-84 fragments, and therefore measure true '**whole** molecule' **PTH**. Transplant recipients (as well as those in end-stage renal failure) have been reported to have elevated levels of 'intact' in comparison with '**whole** molecule' **PTH**. Methods **PTH** concentrations were assessed in serum samples obtained from female renal transplant recipients previously classified as either having hyperparathyroid (n = 14) or adynamic bone disease (n = 14) by transiliac crest bone biopsy. **PTH** was measured as '**whole** molecule' (Scantibodies '**whole** molecule' **PTH**) and 'intact' (DPC Immulite 2000 intact **PTH** and Scantibodies total **PTH**). Results Scantibodies '**whole** molecule' **PTH** (all-subject mean 48.7 ng/L, ± 53.0) were significantly lower than DPC intact (83.5 ng/L, ±88.1; P <= 0.0001) and Scantibodies total **PTH** (80.5 ng/L, ±92.4; P <= 0.0001). However, the differences between the '**whole** molecule' and 'intact' measurements were similar across the two patient groups, and reflected the lower reference range employed by the '**whole**

molecule' assay. Conclusion The '**whole** molecule' **PTH** assay was unable to discriminate between the two patient populations and provided very little additional clinical information to that obtained from the intact **PTH** assays.

L19 ANSWER 4 OF 11 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
DUPLICATE

ACCESSION NUMBER: 2001:32230589 BIOTECHNO
TITLE: Development of a novel immunoradiometric assay exclusively for biologically active **whole parathyroid hormone** 1-84: Implications for improvement of accurate assessment of parathyroid function
AUTHOR: Gao P.; Scheibel S.; D'Amour P.; John M.R.; Rao S.D.; Schmidt-Gayk H.; Cantor T.L.
CORPORATE SOURCE: Dr. P. Gao, Department of R and D, Scantibodies Laboratory, Inc., 9336 Abraham Way, Santee, CA 92071, United States.
SOURCE: Journal of Bone and Mineral Research, (2001), 16/4 (605-614), 27 reference(s)
CODEN: JBMREJ ISSN: 0884-0431
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2001:32230589 BIOTECHNO
AB We developed a novel immunoradiometric assay (IRMA; **whole parathyroid hormone** [**PTH**] IRMA) for **PTH**, which specifically measures biologically active **whole PTH**(1-84). The assay is based on a solid phase coated with anti-**PTH**(39-84) **antibody**, a tracer of .sup.1.sup.2.sup.5I-labeled **antibody** with a unique specificity to the first N-terminal amino acid of **PTH**(1-84), and calibrators of diluted synthetic **PTH**(1-84). In contrast to the Nichols intact **PTH** IRMA, this new assay does not detect **PTH**(7-84) fragments and only detects one immunoreactive peak in chromatographically fractionated patient samples. The assay was shown to have an analytical sensitivity of 1.0 pg/ml with a linear measurement range up to 2300 pg/ml. With this assay, we further identified that the previously described non-(1-84)**PTH** fragments are aminoterminally truncated with similar hydrophobicity as **PTH** (7-84), and these **PTH** fragments are present not only in patients with secondary hyperparathyroidism (2°-HPT) of uremia, but also in patients with primary hyperparathyroidism (1°-HPT) and normal persons. The plasma normal range of the **whole PTH**(1-84) was 7-36 pg/ml (mean ± SD: 22.7 ± 7.2 pg/ml, n = 135), whereas over 93.9% (155/165) of patients with 1°-HPT had **whole PTH**(1-84) values above the normal cut-off. The percentage of biologically active **whole PTH**(1-84) (pB%) in the pool of total immunoreactive "intact" **PTH** is higher in the normal population (median: 67.3%; SD: 15.8%; n = 56) than in uremic patients (median: 53.8%; SD: 15.5%; n = 318; $p < 0.001$), although the **whole PTH**(1-84) values from uremic patients displayed a more significant heterogeneous distribution when compared with that of 1°-HPT patients and normals. Moreover, the pB% displayed a nearly Gaussian distribution pattern from 20% to over 90% in patients with either 1°-HPT or uremia. The specificity of this newly developed **whole PTH**(1-84) IRMA is the assurance, for the first time, of being able to measure only the biologically active **whole PTH**(1-84) without cross-reaction to the high concentrations of the aminoterminally truncated **PTH** fragments found in both normal subjects and patients. Because of the significant variations of pB% in patients, it is necessary to use the **whole PTH** assay to determine

biologically active **PTH** levels clinically and, thus, to avoid overestimating the concentration of the true biologically active hormone. This new assay could provide a more meaningful standardization of future **PTH** measurements with improved accuracy in the clinical assessment of parathyroid function.

L19 ANSWER 5 OF 11 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
DUPLICATE

ACCESSION NUMBER: 2000:30620025 BIOTECHNO
TITLE: A novel mechanism for skeletal resistance in uremia
AUTHOR: Slatopolsky E.; Finch J.; Clay P.; Martin D.; Sicard G.; Singer G.; Gao P.; Cantor T.; Dusso A.
CORPORATE SOURCE: Dr. E. Slatopolsky, Washington Univ. School of Medicine, Renal Division, Box 8126, 660 South Euclid Avenue, St. Louis, MO 63110, United States.
SOURCE: Kidney International, (2000), 58/2 (753-761), 31 reference(s)
CODEN: KDYIA5 ISSN: 0085-2538
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2000:30620025 BIOTECHNO
AB Background. In treating secondary hyperparathyroidism, the target level of serum intact **parathyroid hormone** (I-**PTH**) should be three to five times normal to prevent adynamic bone disease. In circulation, there is a non-(1-84) **PTH**-truncated fragment, likely 7-84, which, in addition to **PTH** 1-84, is measured by most I-**PTH** immunoradiometric (IRMA) assays, giving erroneously high I-**PTH** values. We have developed a new IRMA assay in which the labeled **antibody** recognizes only the first six amino acids of the **PTH** molecule. Thus, this new IRMA assay (**Whole PTH**) measures only the biologically active 1-84 **PTH** molecule. Methods. Using this new IRMA assay (**Whole PTH**) and the Nichols 'intact' **PTH** assay, we compared the ability of each assay to recognize human **PTH** (hPTH) 1-84 and hPTH 7-84 and examined the percentage of non-1-84 **PTH** in circulation and in parathyroid glands. Possible antagonistic effects of the 7-84 **PTH** fragment on the biological activity of 1-84 **PTH** in rats were also tested. Results. In 28 uremic patients, **PTH** values measured with the Nichols assay, representing a combined measurement of both hPTH 1-84 and hPTH 7-84, were 34% higher than with the **Whole** assay (hPTH 1-84 only); the median **PTH** was 523 versus 318 pg/mL ($P < 0.001$). Similar results were found in 14 renal transplant patients. In osteoblast-like cells, ROS 17.2, 1-84 **PTH** (10.sup.-.sup.8 mol/L) increased cAMP from 18.1 ± 1.25 to 738 ± 4.13 mmol/well. Conversely, the same concentration of 7-84 **PTH** had no effect. In parathyroidectomized rats fed a calcium-deficient diet, 7-84 **PTH** was not only biologically inactive, but had antagonistic effects on 1-84 **PTH** in bone. Plasma calcium was increased (0.65 mg/dL) two hours after 1-84 **PTH** treatment, while 7-84 **PTH** had no effect. When 1-84 **PTH** and 7-84 **PTH** were given simultaneously in a 1:1 molar ratio, the calcemic response to 1-84 **PTH** was decreased by 94%. In normal rats, the administration of 1-84 **PTH** increased renal fractional excretion of phosphate (11.9 to 27.7%, $P < 0.001$). However, when 1-84 **PTH** and 7-84 **PTH** were given simultaneously, the 7-84 **PTH** decreased the phosphaturic response by 50.2% ($P < 0.005$). Finally, in surgically excised parathyroid glands from six uremic patients, we found that 44.1% of the total intracellular **PTH** was the non-**PTH** (1-84), most likely **PTH** 7-84. Conclusion. In patients with chronic renal failure, the presence of high circulating levels of non-1-84 **PTH** fragments (most likely 7-84 **PTH**) detected by the 'intact' assay and the antagonistic effects of 7-84

```
Connecting via Winsock to STN



Welcome to STN International!  Enter x:x

LOGINID:ssspta1641cxc

PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2


* * * * * * * * * *   Welcome to STN International   * * * * * * * * * *

 NEWS  1           Web Page URLs for STN Seminar Schedule - N. America
 NEWS  2           "Ask CAS" for self-help around the clock
 NEWS  3  SEP 01   New pricing for the Save Answers for SciFinder Wizard within
                   STN Express with Discover!
 NEWS  4  OCT 28   KOREAPAT now available on STN
 NEWS  5  NOV 30   PHAR reloaded with additional data
 NEWS  6  DEC 01   LISA now available on STN
 NEWS  7  DEC 09   12 databases to be removed from STN on December 31, 2004
 NEWS  8  DEC 15   MEDLINE update schedule for December 2004
 NEWS  9  DEC 17   ELCOM reloaded; updating to resume; current-awareness
                   alerts (SDIs) affected
 NEWS  10 DEC 17   COMPUAB reloaded; updating to resume; current-awareness
                   alerts (SDIs) affected
 NEWS  11 DEC 17   SOLIDSTATE reloaded; updating to resume; current-awareness
                   alerts (SDIs) affected
 NEWS  12 DEC 17   CERAB reloaded; updating to resume; current-awareness
                   alerts (SDIs) affected
 NEWS  13 DEC 17   THREE NEW FIELDS ADDED TO IFIPAT/IFIUDB/IFICDB
 NEWS  14 DEC 30   EPFULL: New patent full text database to be available on STN
 NEWS  15 DEC 30   CAPLUS - PATENT COVERAGE EXPANDED

 NEWS EXPRESS  OCTOBER 29 CURRENT WINDOWS VERSION IS V7.01A, CURRENT
               MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
               AND CURRENT DISCOVER FILE IS DATED 11 AUGUST 2004
 NEWS HOURS    STN Operating Hours Plus Help Desk Availability
 NEWS INTER    General Internet Information
 NEWS LOGIN    Welcome Banner and News Items
 NEWS PHONE    Direct Dial and Telecommunication Network Access to STN
 NEWS WWW      CAS World Wide Web Site (general information)

Enter NEWS followed by the item number or name to see news on that
specific topic.

  All use of STN is subject to the provisions of the STN Customer
  agreement.  Please note that this agreement limits use to scientific
  research.  Use for software development or design or implementation
  of commercial gateways or other similar uses is prohibited and may
  result in loss of user privileges and other penalties.

  * * * * * * * * * * * * * * STN  Columbus  * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 16:05:37 ON 03 JAN 2005

=> file .meeting
'EVENTLINE' IS NOT A VALID FILE NAME
Enter "HELP FILE NAMES" at an arrow prompt (=>) for a list of files
that are available.  If you have requested multiple files, you can
specify a corrected file name or you can enter "IGNORE" to continue
```

```
accessing the remaining file names entered.
ENTER A FILE NAME OR (IGNORE):ignore
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                        ENTRY    SESSION
FULL ESTIMATED COST                                      0.21       0.21

FILE 'AGRICOLA' ENTERED AT 16:05:52 ON 03 JAN 2005

FILE 'BIOTECHNO' ENTERED AT 16:05:52 ON 03 JAN 2005
COPYRIGHT (C) 2005 Elsevier Science B.V., Amsterdam. All rights reserved.

FILE 'CONFSCI' ENTERED AT 16:05:52 ON 03 JAN 2005
COPYRIGHT (C) 2005 Cambridge Scientific Abstracts (CSA)

FILE 'HEALSAFE' ENTERED AT 16:05:52 ON 03 JAN 2005
COPYRIGHT (C) 2005 Cambridge Scientific Abstracts (CSA)


FILE 'IMSDRUGCONF' ENTERED AT 16:05:52 ON 03 JAN 2005
COPYRIGHT (C) 2005 IMSWORLD Publications Ltd.

FILE 'LIFESCI' ENTERED AT 16:05:52 ON 03 JAN 2005
COPYRIGHT (C) 2005 Cambridge Scientific Abstracts (CSA)


FILE 'MEDICONF' ENTERED AT 16:05:52 ON 03 JAN 2005
COPYRIGHT (c) 2005 FAIRBASE Datenbank GmbH, Hannover, Germany

FILE 'PASCAL' ENTERED AT 16:05:52 ON 03 JAN 2005
Any reproduction or dissemination in part or in full,
by means of any process and on any support whatsoever
is prohibited without the prior written agreement of INIST-CNRS.
COPYRIGHT (C) 2005 INIST-CNRS. All rights reserved.

=> antibody and (pth or parathyroid hormone) and whole and non-whole
L1            0 FILE AGRICOLA
L2            0 FILE BIOTECHNO
L3            0 FILE CONFSCI
L4            0 FILE HEALSAFE
L5            0 FILE IMSDRUGCONF
L6            0 FILE LIFESCI
L7            0 FILE MEDICONF
L8            0 FILE PASCAL

TOTAL FOR ALL FILES
L9            0 ANTIBODY AND (PTH OR PARATHYROID HORMONE) AND WHOLE AND NON-WHOL
                E

=> antibody and (pth or parathyroid hormone) and whole
L10           0 FILE AGRICOLA
L11           9 FILE BIOTECHNO
L12           0 FILE CONFSCI
L13           0 FILE HEALSAFE
L14           0 FILE IMSDRUGCONF
L15           3 FILE LIFESCI
L16           0 FILE MEDICONF
L17           7 FILE PASCAL

TOTAL FOR ALL FILES
L18          19 ANTIBODY AND (PTH OR PARATHYROID HORMONE) AND WHOLE

=> dup rem
ENTER L# LIST OR (END):l19
L19 IS NOT VALID HERE
```

The L-number entered has not been defined in this session, or it has been deleted. To see the L-numbers currently defined in this session, enter DISPLAY HISTORY at an arrow prompt (=>).

=> dup rem
ENTER L# LIST OR (END):l18
DUPLICATE IS NOT AVAILABLE IN 'IMSDRUGCONF, MEDICONF'.
ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE
PROCESSING COMPLETED FOR L18
L19 11 DUP REM L18 (8 DUPLICATES REMOVED)

=> d l19 ibib abs total

L19 ANSWER 1 OF 11 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
DUPLICATE
ACCESSION NUMBER: 2003:36432259 BIOTECHNO
TITLE: Influence of **PTH** assay methodology on differential diagnosis of renal bone disease
AUTHOR: Reichel H.; Esser A.; Roth H.-J.; Schmidt-Gayk H.
CORPORATE SOURCE: Dr. H. Reichel, Nephrological Center, Schramberger Strasse 28, D-78054 Villingen-Schwenningen, Germany. E-mail: helmut.reichel@dialyse-schwenningen.de
SOURCE: Nephrology Dialysis Transplantation, (01 APR 2003), 18/4 (759-768), 20 reference(s)
CODEN: NDTREA ISSN: 0931-0509
DOCUMENT TYPE: Journal; Article
COUNTRY: United Kingdom
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2003:36432259 BIOTECHNO
AB Background. Determination of plasma **parathyroid hormone** (**PTH**) is routinely performed to diagnose and monitor renal bone disease. Recently, a new **PTH** assay ('**whole PTH**') using an **antibody** directed specifically against **PTH**(1-4) has been introduced. It was the aim of the current study to evaluate **whole PTH** and parameters derived from **whole PTH** in renal bone disease. Methods. The following measurements were carried out in blood samples from 141 unselected haemodialysis patients: three intact **PTH** assays (Nichols, Roche Elecsys®, Scantibodies total); **whole PTH** (Scantibodies); bone-specific alkaline phosphatase (bAP); tartrate-resistant acid-phosphatase 5b (TRAP 5b); osteocalcin, 25-hydroxyvitamin D. Parameters derived from **whole PTH** were: (i) non-**PTH**(1-84), difference between intact **PTH** (Scantibodies assay) and **whole PTH**; (ii) **whole PTH**/non-**PTH**(1-84) ratio. Results. The values generated by the intact **PTH** assays were comparable. The mean **whole PTH** concentration was lower than mean intact **PTH** concentrations (16.9 ± 18.1 vs 26.4 ± 30.5 pmol/l, Nichols, $P < 0.05$). The correlation coefficients between all four **PTH** assays were comparable and were very high ($r > 0.96$, ns). The rank order of values generated by the **whole PTH** assay was statistically not significantly different from the rank order generated by the Nichols intact **PTH** assay. The median non-**PTH**(1-84) concentration was 5.2 pmol/l (range 0-49.4). All **PTH** assays correlated highly significantly with non-**PTH** (1-84) (correlation coefficients 0.83-0.92). Corrected serum calcium was also associated with non-**PTH** (1-84) but the correlation was weaker ($r = 0.28$). Regression analysis indicated that the non-**PTH** (1-84) concentration could be predicted by 76.6-84.6% by the prevailing intact **PTH** concentrations. Other parameters contributed only marginally to prediction of non-**PTH**(1-84). In the entire patient group, there was no statistically significant correlation between the **whole PTH**/non-**PTH**(1-84) ratio and any

of the **PTH** assays or biochemical bone markers. Eight of 141 patients had a **whole PTH**/non-**PTH**(1-84) ratio < 1. TRAP 5b, bAP and osteocalcin had high correlations with intact **PTH** assays and the **whole PTH** assay (correlation coefficients 0.51-0.56, no significant difference). None of the **PTH** assays was superior to any other **PTH** assay in predicting serum concentrations of the bone markers. Therapy with active vitamin D metabolites (n = 70) did not alter the results of our analyses. Conclusions. With respect to information about bone turnover we were not able to find differences between **whole PTH** and intact **PTH** assays. Our data also suggest that **whole PTH** and intact **PTH** assays give similar information. (i) The correlation between all **PTH** assays was very high. (ii) The rank order between **whole PTH** and Nichols intact **PTH** assays was comparable. (iii) The association between intact **PTH** assays and non-**PTH**(1-84) was very high. Albeit non-**PTH**(1-84) was mostly determined by the prevailing intact **PTH** concentration, diagnostic information on parathyroid activity provided by **whole PTH** or intact **PTH**, respectively, may differ in individual patients. How often this would happen cannot be answered with the currently available data. Unequivocal structural identification of the non-**PTH**(1-84) fraction would facilitate the answer to that question. The use of the **whole PTH**/non-**PTH**(1-84) ratio as a biochemical bone marker in renal bone disease requires further investigation.

L19 ANSWER 2 OF 11 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
DUPLICATE

ACCESSION NUMBER: 2003:36592205 BIOTECHNO
TITLE: **Parathyroid hormone** assays - Evolution and revolutions in the care of dialysis patients
AUTHOR: Malluche H.H.; Mawad H.; Trueba D.; Monier-Faugere M.-C.
CORPORATE SOURCE: Dr. H.H. Malluche, Div Nephrology Bone/Mineral Metab., UK Medical Center, University of Kentucky, 800 Rose Street, Lexington, KY 40536-0084, United States. E-mail: hhmall@pop.uky.edu
SOURCE: Clinical Nephrology, (01 MAY 2003), 59/5 (313-318), 45 reference(s)
CODEN: CLNHBI ISSN: 0301-0430
DOCUMENT TYPE: Journal; General Review
COUNTRY: Germany, Federal Republic of
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2003:36592205 BIOTECHNO
AB Renal osteodystrophy may present with low, normal, or high bone turnover. An ideal **parathyroid hormone** (**PTH**) assay should discriminate between the bioactive **whole PTH** -(1-84) molecule and **PTH** fragments, including the **PTH** -(7-84) fragment. Most dialysis patients have "intact" **PTH** (iPTH) levels between 65 and 450 pg/ml, which are poorly predictive of bone turnover state, making the iPTH test of limited value for bone turnover prediction. iPTH levels higher than 500 pg/ml can be observed in some dialysis patients with low bone turnover, while iPTH levels as low as 100 pg/ml have been found in patients with bone turnover above normal, indicating the standard second generation iPTH assay is not a reliable sole indicator of bone turnover. The **whole PTH** immunoradiometric assay, a third generation assay, uses a detection **antibody** that recognizes antigenic determinants at the extreme amino-terminal (1-4) end of the **PTH** molecule, making the assay specific for biologically active **whole PTH**-(1-84). Comparing results using the **whole PTH** and iPTH assays, the **PTH**-(7-84) level is indirectly determined and the

PTH-(1-84)/**PTH**-(7-84) ratio can be calculated. It was shown that **PTH**-(7-84) inhibits the calcemic effect of **PTH**-(1-84) and its stimulatory effect on bone turnover. In the interpretation of results using the **PTH**-(1-84)/**PTH**-(7-84) ratio, it must be taken into consideration that second generation "intact" **PTH** assays have different cross-reactivity with **PTH**-(7-84). Therefore, when comparing or analyzing **PTH**-(1-84)/**PTH**-(7-84) ratios, the employed **PTH** assays must be identical. The **whole PTH** assay and the **PTH**-(1-84)/**PTH**-(7-84) ratio allow more meaningful interpretation of **PTH** trends, and offer a noninvasive means to more accurately diagnose bone disease in this population.

L19 ANSWER 3 OF 11 PASCAL COPYRIGHT 2005 INIST-CNRS. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 2003-0047687 PASCAL
COPYRIGHT NOTICE: Copyright .COPYRGT. 2003 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): Comparison of intact and '**whole** molecule' **parathyroid hormone** assays in patients with histologically confirmed post-renal transplant osteodystrophy
AUTHOR: GODBER Ian M.; PARKER Cornelle R.; LAWSON Nigel; HITCH Tony; PORTER Christine J.; ROE Simon D.; CASSIDY Michael J. D.; HOSKING David J.
CORPORATE SOURCE: Department of Clinical Chemistry, Nottingham City Hospital, Hucknall Road, Nottingham NG5 1PB, United Kingdom; Division of Mineral Metabolism, Nottingham City Hospital, Hucknall Road, Nottingham NG5 1PB, United Kingdom; Nottingham Renal Unit, Nottingham City Hospital Hucknall Road, Nottingham NG5 1PB, United Kingdom
SOURCE: Annals of clinical biochemistry, (2002), 39(p.3), 314-317, 8 refs.
ISSN: 0004-5632 CODEN: ACBOBU
DOCUMENT TYPE: Journal; Short communication
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United Kingdom
LANGUAGE: English
AVAILABILITY: INIST-17507, 354000104964540200
AN 2003-0047687 PASCAL
CP Copyright .COPYRGT. 2003 INIST-CNRS. All rights reserved.
AB Background Fragments of **parathyroid hormone** (**PTH**) have been identified (amino acids 7-84) which may interfere with commercially available 'intact molecule' **PTH** assays. Novel assays which employ an **antibody** directed to the first seven amino acids of the N-terminus of **PTH** are thought to be free from cross-reactivity with the 7-84 fragments, and therefore measure true '**whole** molecule' **PTH**. Transplant recipients (as well as those in end-stage renal failure) have been reported to have elevated levels of 'intact' in comparison with '**whole** molecule' **PTH**. Methods **PTH** concentrations were assessed in serum samples obtained from female renal transplant recipients previously classified as either having hyperparathyroid (n = 14) or adynamic bone disease (n = 14) by transiliac crest bone biopsy. **PTH** was measured as '**whole** molecule' (Scantibodies '**whole** molecule' **PTH**) and 'intact' (DPC Immulite 2000 intact **PTH** and Scantibodies total **PTH**). Results Scantibodies '**whole** molecule' **PTH** (all-subject mean 48.7 ng/L, ± 53.0) were significantly lower than DPC intact (83.5 ng/L, ±88.1; P <= 0.0001) and Scantibodies total **PTH** (80.5 ng/L, ±92.4; P <= 0.0001). However, the differences between the '**whole** molecule' and 'intact' measurements were similar across the two patient groups, and reflected the lower reference range employed by the '**whole**

molecule' assay. Conclusion The '**whole** molecule' **PTH** assay was unable to discriminate between the two patient populations and provided very little additional clinical information to that obtained from the intact **PTH** assays.

L19 ANSWER 4 OF 11 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
DUPLICATE

ACCESSION NUMBER: 2001:32230589 BIOTECHNO
TITLE: Development of a novel immunoradiometric assay exclusively for biologically active **whole parathyroid hormone** 1-84: Implications for improvement of accurate assessment of parathyroid function
AUTHOR: Gao P.; Scheibel S.; D'Amour P.; John M.R.; Rao S.D.; Schmidt-Gayk H.; Cantor T.L.
CORPORATE SOURCE: Dr. P. Gao, Department of R and D, Scantibodies Laboratory, Inc., 9336 Abraham Way, Santee, CA 92071, United States.
SOURCE: Journal of Bone and Mineral Research, (2001), 16/4 (605-614), 27 reference(s)
CODEN: JBMREJ ISSN: 0884-0431
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2001:32230589 BIOTECHNO
AB We developed a novel immunoradiometric assay (IRMA; **whole parathyroid hormone** [**PTH**] IRMA) for **PTH**, which specifically measures biologically active **whole PTH**(1-84). The assay is based on a solid phase coated with anti-**PTH**(39-84) **antibody**, a tracer of .sup.1.sup.2.sup.5I-labeled **antibody** with a unique specificity to the first N-terminal amino acid of **PTH**(1-84), and calibrators of diluted synthetic **PTH**(1-84). In contrast to the Nichols intact **PTH** IRMA, this new assay does not detect **PTH**(7-84) fragments and only detects one immunoreactive peak in chromatographically fractionated patient samples. The assay was shown to have an analytical sensitivity of 1.0 pg/ml with a linear measurement range up to 2300 pg/ml. With this assay, we further identified that the previously described non-(1-84)**PTH** fragments are aminoterminally truncated with similar hydrophobicity as **PTH** (7-84), and these **PTH** fragments are present not only in patients with secondary hyperparathyroidism (2°-HPT) of uremia, but also in patients with primary hyperparathyroidism (1°-HPT) and normal persons. The plasma normal range of the **whole PTH**(1-84) was 7-36 pg/ml (mean ± SD: 22.7 ± 7.2 pg/ml, n = 135), whereas over 93.9% (155/165) of patients with 1°-HPT had **whole PTH**(1-84) values above the normal cut-off. The percentage of biologically active **whole PTH**(1-84) (pB%) in the pool of total immunoreactive "intact" **PTH** is higher in the normal population (median: 67.3%; SD: 15.8%; n = 56) than in uremic patients (median: 53.8%; SD: 15.5%; n = 318; $p < 0.001$), although the **whole PTH**(1-84) values from uremic patients displayed a more significant heterogeneous distribution when compared with that of 1°-HPT patients and normals. Moreover, the pB% displayed a nearly Gaussian distribution pattern from 20% to over 90% in patients with either 1°-HPT or uremia. The specificity of this newly developed **whole PTH**(1-84) IRMA is the assurance, for the first time, of being able to measure only the biologically active **whole PTH**(1-84) without cross-reaction to the high concentrations of the aminoterminally truncated **PTH** fragments found in both normal subjects and patients. Because of the significant variations of pB% in patients, it is necessary to use the **whole PTH** assay to determine

biologically active **PTH** levels clinically and, thus, to avoid overestimating the concentration of the true biologically active hormone. This new assay could provide a more meaningful standardization of future **PTH** measurements with improved accuracy in the clinical assessment of parathyroid function.

L19 ANSWER 5 OF 11 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
DUPLICATE

ACCESSION NUMBER: 2000:30620025 BIOTECHNO
TITLE: A novel mechanism for skeletal resistance in uremia
AUTHOR: Slatopolsky E.; Finch J.; Clay P.; Martin D.; Sicard G.; Singer G.; Gao P.; Cantor T.; Dusso A.
CORPORATE SOURCE: Dr. E. Slatopolsky, Washington Univ. School of Medicine, Renal Division, Box 8126, 660 South Euclid Avenue, St. Louis, MO 63110, United States.
SOURCE: Kidney International, (2000), 58/2 (753-761), 31 reference(s)
CODEN: KDYIA5 ISSN: 0085-2538
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2000:30620025 BIOTECHNO
AB Background. In treating secondary hyperparathyroidism, the target level of serum intact **parathyroid hormone** (I-**PTH**) should be three to five times normal to prevent adynamic bone disease. In circulation, there is a non-(1-84) **PTH**-truncated fragment, likely 7-84, which, in addition to **PTH** 1-84, is measured by most I-**PTH** immunoradiometric (IRMA) assays, giving erroneously high I-**PTH** values. We have developed a new IRMA assay in which the labeled **antibody** recognizes only the first six amino acids of the **PTH** molecule. Thus, this new IRMA assay (**Whole PTH**) measures only the biologically active 1-84 **PTH** molecule. Methods. Using this new IRMA assay (**Whole PTH**) and the Nichols 'intact' **PTH** assay, we compared the ability of each assay to recognize human **PTH** (hPTH) 1-84 and hPTH 7-84 and examined the percentage of non-1-84 **PTH** in circulation and in parathyroid glands. Possible antagonistic effects of the 7-84 **PTH** fragment on the biological activity of 1-84 **PTH** in rats were also tested. Results. In 28 uremic patients, **PTH** values measured with the Nichols assay, representing a combined measurement of both hPTH 1-84 and hPTH 7-84, were 34% higher than with the **Whole** assay (hPTH 1-84 only); the median **PTH** was 523 versus 318 pg/mL ($P < 0.001$). Similar results were found in 14 renal transplant patients. In osteoblast-like cells, ROS 17.2, 1-84 **PTH** (10.sup.-.sup.8 mol/L) increased cAMP from 18.1 ± 1.25 to 738 ± 4.13 mmol/well. Conversely, the same concentration of 7-84 **PTH** had no effect. In parathyroidectomized rats fed a calcium-deficient diet, 7-84 **PTH** was not only biologically inactive, but had antagonistic effects on 1-84 **PTH** in bone. Plasma calcium was increased (0.65 mg/dL) two hours after 1-84 **PTH** treatment, while 7-84 **PTH** had no effect. When 1-84 **PTH** and 7-84 **PTH** were given simultaneously in a 1:1 molar ratio, the calcemic response to 1-84 **PTH** was decreased by 94%. In normal rats, the administration of 1-84 **PTH** increased renal fractional excretion of phosphate (11.9 to 27.7%, $P < 0.001$). However, when 1-84 **PTH** and 7-84 **PTH** were given simultaneously, the 7-84 **PTH** decreased the phosphaturic response by 50.2% ($P < 0.005$). Finally, in surgically excised parathyroid glands from six uremic patients, we found that 44.1% of the total intracellular **PTH** was the non-**PTH** (1-84), most likely **PTH** 7-84. Conclusion. In patients with chronic renal failure, the presence of high circulating levels of non-1-84 **PTH** fragments (most likely 7-84 **PTH**) detected by the 'intact' assay and the antagonistic effects of 7-84

PTH on the biological activity of 1-84 **PTH** explain the need of higher levels of 'intact' **PTH** to prevent adynamic bone disease.

L19 ANSWER 6 OF 11 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
DUPLICATE
ACCESSION NUMBER: 1997:27329806 BIOTECHNO
TITLE: History of posttransfusion hepatitis
AUTHOR: Tobler L.H.; Busch M.P.
CORPORATE SOURCE: L.H. Tobler, Irwin Memorial Blood Centers, 270 Masonic Ave, San Francisco, CA 94118, United States.
E-mail: itobler@ccnet.com
SOURCE: Clinical Chemistry, (1997), 43/8 SUPPL. (1487-1493), 47 reference(s)
CODEN: CLCHAU ISSN: 0009-9147
DOCUMENT TYPE: Journal; Conference Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 1997:27329806 BIOTECHNO
AB The risk of hepatitis virus transmission from transfusions has declined dramatically from that of the 1940s when posttransfusion hepatitis (**PTH**) was first appreciated. Introduction of hepatitis B surface antigen screening and conversion to volunteer donors for **whole** -blood donations in the late 1960s and early 1970s led to substantial reduction in **PTH** cases. However, up to 10% of the recipients continued to develop **PTH**, most cases of which were attributed to an unknown non-A, non-B vital agent. Implementation of surrogate marker testing (i.e., alanine aminotransferase and anti-hepatitis B virus core antigen) for residual non-A, non-B hepatitis in the late 1980s reduced the per unit risk of **PTH** from 1 in 200 to about 1 in 400. Hepatitis C virus was discovered in 1989 and quickly was established as the causative agent of >90% of non-A, non-B **PTH**. Introduction of progressively improved **antibody** assays in the early 1990s reduced the risk of **PTH** due to hepatitis C virus to about 1 in 100 000. Although additional hepatitis viruses exist (e.g., hepatitis G virus), these appear to be minor contributors to clinical **PTH**, which has been virtually eradicated.

L19 ANSWER 7 OF 11 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
DUPLICATE
ACCESSION NUMBER: 1996:26330992 BIOTECHNO
TITLE: **PTH**-related protein is released into the mother's bloodstream during lactation: Evidence for beneficial effects on maternal calcium-phosphate metabolism
AUTHOR: Lippuner K.; Zehnder H.-J.; Casez J.-P.; Takkinen R.; Jaeger P.
CORPORATE SOURCE: Policlinic of Medicine, University Hospital,3010 Berne, Switzerland.
SOURCE: Journal of Bone and Mineral Research, (1996), 11/10 (1394-1399)
CODEN: JBMREJ ISSN: 0884-0431
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 1996:26330992 BIOTECHNO
AB Recent studies have indicated that **parathyroid hormone** -related protein (PTHrP) may have important actions in lactation, affecting the mammary gland, and also calcium metabolism in the newborn and the mother. However, there are as yet no longitudinal studies to support the notion of an endocrine role of this peptide during nursing. We studied a group of 12 nursing mothers, mean age 32 years, after they

had been nursing for an average of 7 weeks (B) and also 4 months after stopping nursing (A). It was assumed that changes occurring between A and B correspond to the effect of lactation. Blood was assayed for prolactin (PRL), PTHrP (two-site immunoradiometric assay with sheep **antibody** against PTHrP(1-40), and goat **antibody** against PTHrP(60-72), detection limit 0.3 pmol/l), intact **PTH** (iPTH), ionized calcium (Ca.sup.2.sup.+), 25- hydroxyvitamin D.sub.3 (25(OH)D.sub.3) and 1,25-dihydroxyvitamin D.sub.3 (1,25(OH).sub.2D.sub.3), alkaline phosphatase (alkP), as well as for creatinine (Cr), protein, phosphorus (P), and total calcium (Ca). Fasting 2-h urine samples were analyzed for Ca excretion (CaE) and renal phosphate threshold (TmP/GFR). PRL was significantly higher during lactation than after weaning (39 ± 10 vs. 13 ± 9 μg/l; p = 0.018) and so was PTHrP (2.8 ± 0.35 vs. 0.52 ± 0.04 pmol/l; p = 0.002), values during lactation being above the normal limit (1.3 pmol/l) in all 12 mothers. There was a significant correlation between PRL and PTHrP during lactation (r = 0.8, p = 0.002). **Whole** blood Ca.sup.2.sup.+ did not significantly change from A (1.20 ± 0.02 mmol/l) to B (1.22 ± 0.02, mmol/l), whereas total Ca corrected for protein (2.18 ± 0.02 mmol/l) or uncorrected (2.18 ± 0.02 mmol/l) significantly rose during lactation (2.31 ± 0.02 mmol/l, p = 0.003 and 2.37 ± 0.03 mmol/l, p = 0.002, respectively). Conversely, iPTH decreased during lactation (3.47 ± 0.38 vs. 2.11 ± 0.35 pmol/l, A vs. B, p = 0.02). Serum-levels of 25(OH)D.sub.3 and 1,25(OH).sub.2D.sub.3 did not significantly change from A to B (23 ± 2.3 vs. 24 ± 1.9 ng/ml and 29.5 ± 6.0 vs. 21.9 ± 1.8 pg/ml, respectively). Both TmP/GFR and P were higher during lactation than after weaning (1.15 ± 0.03 vs. 0.86 ± 0.05 mmol/l GF, p = 0.003 and 1.25 ± 0.03 vs. 0.96 ± 0.05 mmol/l, p = 0.002, respectively) as was alkP (74.0 ± 7.1 vs. 52.6 ± 6.9 U/l, p = 0.003). CaE did not differ between A and B (0.015 ± 0.003 vs. 0.017 ± 0.003 mmol/l GF, A vs. B, NS). We conclude that lactation is accompanied by an increase in serum PRL. This is associated with a release of PTHrP into the maternal blood circulation. A rise in total plasma Ca ensues, probably in part by increased bone turnover as suggested by the elevation of alkP. **PTH** secretion falls, with a subsequent rise of TmP/GFR and plasma P despite high plasma levels of PTHrP.

L19 ANSWER 8 OF 11 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
ACCESSION NUMBER: 1991:21314326 BIOTECHNO
TITLE: Incidence of Anti-Hepatitis C virus **antibodies** in Non-A, Non-B Post-Transfusion hepatitis in an area of Northern Italy
AUTHOR: Elia G.F.; Magnani G.; Belli L.; Formentini A.; Iacono S.; Marchelli Pincolini S.S.; Lecchini R.; Bernuzzi G.; Fiaccadori F.
CORPORATE SOURCE: Universita' di Parma, Servizio Anestesia, Ospedsle Maggiore di Parma, Via A Gramsci 14,I-43100 Parma, Italy.
SOURCE: Infection, (1991), 19/5 (336-339)
CODEN: IFTNAL ISSN: 0300-8126
DOCUMENT TYPE: Journal; Article
COUNTRY: Germany, Federal Republic of
LANGUAGE: English
SUMMARY LANGUAGE: German; English
AN 1991:21314326 BIOTECHNO
AB A total of 210 patients consecutively submitted to heart surgery at the Parma University Hospital and transfused with 1,898 units of blood were followed after transfusion in order to evaluate both the incidence of anti-hepatitis C virus (HCV) seroconversion in non-A, non-B post-transfusion hepatitis (**PTH**-NANB) cases and the usefulness of the screening for anti-HCV in comparison with that for serum glutamic pyruvic transaminase (SGPT) values in preventing **PTH**-NANB transmission. Fifteen recipients developed **PTH**-NANB (group A);

ten of them (66.6%) showed anti-HCV seroconversion within 3-12 months. Eight of the ten anti-HCV positive patients developed chronic hepatitis, but none of the five **PTH**-NANB anti-HCV negative did. None of the 15 controls (group B) randomly chosen among the patient population showed anti-HCV seroconversion. A close correlation with the transmission of **PTH** was showed by anti-HCV positivity but not by SGPT elevation in blood donors. Eleven of 172 blood products transfused to group A but none of 139 products transfused to group B were anti-HCV positive. The incidence of elevated SGPT values was similar between the two groups of the transfused blood products. Nevertheless, the correlation observed between anti-HCV positivity and SGPT levels in the blood products involved in **PTH** confirms the need to exclude blood donors with abnormal SGPT values. On the **whole**, anti-HCV screening of donors showed a predictive value higher than that of SGPT (100% vs. 53.3%), allowing a minor blood donation exclusion. The percentage of anti-HCV seroconversion observed in **PTH**-NANB is probably underestimated because of the limits of the ELISA method we used for the detection of anti-HCV.

L19 ANSWER 9 OF 11 LIFESCI COPYRIGHT 2005 CSA on STN
ACCESSION NUMBER: 85:76395 LIFESCI
TITLE: Localization of **parathyroid hormone** -like substance in squamous cell carinomas: An immunoperoxidase study with ultrastructural correlation.
AUTHOR: Ilardi, C.F.; Faro, J.C.
CORPORATE SOURCE: Dep. Lab., Long Island Jewish Med. Cent., New Hyde Park, NY 11042, USA
SOURCE: ARCH. PATHOL. LAB. MED., (1985) vol. 109, no. 8, pp. 752-755.
DOCUMENT TYPE: Journal
FILE SEGMENT: T
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Histologic sections of squamous cell carcinomas of four hypercalcemic patients were investigated for the presence of **parathyroid hormone** (**PTH**)-like substance. The Sternberger-peroxidase-antiperoxidase-immunoperoxidase technique utilizing monospecific **antibody** to **whole** (1 to 84) bovine **PTH** demonstrated immunoreactive material in all cases. Electron microscopy of the four tumors revealed dense-core secretory granules resembling those seen in parathyroid chief-cell adenomas. The hypercalcemia associated with some nonmetastatic squamous cell carcinomas is associated with the production of **PTH**-like substance.

L19 ANSWER 10 OF 11 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
ACCESSION NUMBER: 1982:13254064 BIOTECHNO
TITLE: **Parathyroid hormone** assay
AUTHOR: Chih Kao P.
CORPORATE SOURCE: Dep. Lab. Med., Mayo Clin., Rochester, MN, United States.
SOURCE: Mayo Clinic Proceedings, (1982), 57/9 (596-597) CODEN: MACPAJ
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
AN 1982:13254064 BIOTECHNO
AB Antiserum GP-235 recognizes intact **whole**-molecule **PTH** and C-terminal fragments of 44-68 and 53-84 amino acid sequences but does not recognize the N-terminal 1-34 amino acid sequence. A radioimmunoassay was developed with this antiserum, and 118 normal subjects of both sexes from 20 to 55 years of age were studied. The serum **PTH** concentrations in all of these subjects were less than 70 μleq/ml, which is our upper limit of normal. The radioimmunoassay developed with this antiserum can differentiate normal subjects from patients with

primary hyperparathyroidism, hypoparathyroidism, chronic renal failure, or hypercalcemia caused by malignancy.

L19 ANSWER 11 OF 11 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
ACCESSION NUMBER: 1981:12196086 BIOTECHNO
TITLE: Functional receptors for epidermal growth factor on human osteosarcoma cells
AUTHOR: Shupnik M.A.; Tashjian Jr. A.H.
CORPORATE SOURCE: Lab. Toxicol., Harvard Sch. Pub. Hlth, Harvard Med. Sch., Boston, MA 02115, United States.
SOURCE: Journal of Cellular Physiology, (1981), 109/3 (403-410)
CODEN: JCLLAX
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
AN 1981:12196086 BIOTECHNO
AB Previous studies have shown that epidermal growth factor (EGF) stimulates bone resorption in organ culture via a prostaglandin-mediated pathway, and that there are specific receptors for EGF on mouse bone (Tashjian and Levine, '78; Shupnik et al., '80). The present study demonstrates that a clonal line of human osteosarcoma cells, G-292 (Clone A141B1) has specific, high-affinity receptors for EGF and responds to treatment with EGF by increased prostaglandin production. Binding of .sup.1.sup.2.sup.5I-EGF to G-292 cells exhibited a prolonged plateau phase (6 hours); thereafter, binding slowly declined (t.half. = 6 hours) to 30-40% of the maximal level. This decrease in cell-associated .sup.1.sup.2.sup.5I-EGF was prevented by leupeptin (50 μg/ml). EGF binding was of high affinity (K(d) = 1.9 X 10.sup.-.sup.9 M) and was to a single class of non-interacting binding sites. Pretreatment of cells for 48 hours with EGF caused a maximum threefold increase in PGE.sub.2 production, with a half-maximal response at 9.8 X 10.sup.-.sup.1.sup.0 M EGF. Increased PGE.sub.2 production was detectable within 2 hours and the constant presence of EGF was needed to maintain the response. Although EGF is mitogenic in several other systems, it did not increase DNA synthesis in the osteosarcoma cells. EGF treatment also did not increase medium or intracellular cyclic AMP in these cells, although **parathyroid hormone** and exogenous PGE.sub.2 (200 ng/ml) increased cyclic AMP three- to tenfold over control levels. Pretreatment with EGF decreased the level of subsequent .sup.1.sup.2.sup.5I-EGF binding; receptor number decreased to 30-40% of control after 48 hours of treatment, and the half-maximal effect occurred with pretreatment concentrations of 1.6 X 10.sup.-.sup.9 M EGF. In all respects tested, the binding and biological actions of EGF on the human osteosarcoma cells were the same as those in **whole** mouse bone. G-292 cells thus provide a convenient model system to study EGF action on osseous tissue.

=>
<------User Break------>

=> file .meeting
'EVENTLINE' IS NOT A VALID FILE NAME
Enter "HELP FILE NAMES" at an arrow prompt (=>) for a list of files that are available. If you have requested multiple files, you can specify a corrected file name or you can enter "IGNORE" to continue accessing the remaining file names entered.
ENTER A FILE NAME OR (IGNORE):ignore

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	58.03	58.24

FILE 'AGRICOLA' ENTERED AT 16:26:07 ON 03 JAN 2005

FILE 'BIOTECHNO' ENTERED AT 16:26:07 ON 03 JAN 2005

FILE 'CONFSCI' ENTERED AT 16:26:07 ON 03 JAN 2005

FILE 'HEALSAFE' ENTERED AT 16:26:07 ON 03 JAN 2005

FILE 'IMSDRUGCONF' ENTERED AT 16:26:07 ON 03 JAN 2005

FILE 'LIFESCI' ENTERED AT 16:26:07 ON 03 JAN 2005

FILE 'MEDICONF' ENTERED AT 16:26:07 ON 03 JAN 2005

FILE 'PASCAL' ENTERED AT 16:26:07 ON 03 JAN 2005

=> thomase c/au
L20 0 FILE AGRICOLA
L21 0 FILE BIOTECHNO
L22 0 FILE CONFSCI
L23 0 FILE HEALSAFE
'AU' IS NOT A VALID FIELD CODE
L24 0 FILE IMSDRUGCONF
L25 0 FILE LIFESCI
'AU' IS NOT A VALID FIELD CODE
L26 0 FILE MEDICONF
L27 0 FILE PASCAL

TOTAL FOR ALL FILES
L28 0 THOMASE C/AU

=> cantor t/au
L29 1 FILE AGRICOLA
L30 10 FILE BIOTECHNO
L31 1 FILE CONFSCI
L32 0 FILE HEALSAFE
'AU' IS NOT A VALID FIELD CODE
L33 0 FILE IMSDRUGCONF
L34 0 FILE LIFESCI
'AU' IS NOT A VALID FIELD CODE
L35 0 FILE MEDICONF
L36 3 FILE PASCAL

TOTAL FOR ALL FILES
L37 15 CANTOR T/AU

=> l37 and whole PTH
L38 0 FILE AGRICOLA
L39 3 FILE BIOTECHNO
L40 0 FILE CONFSCI
L41 0 FILE HEALSAFE
L42 0 FILE IMSDRUGCONF
L43 0 FILE LIFESCI
L44 0 FILE MEDICONF

L45 1 FILE PASCAL

TOTAL FOR ALL FILES
L46 4 L37 AND WHOLE PTH

=> dup rem
ENTER L# LIST OR (END):l46
DUPLICATE IS NOT AVAILABLE IN 'IMSDRUGCONF, MEDICONF'.
ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE
PROCESSING COMPLETED FOR L46
L47 3 DUP REM L46 (1 DUPLICATE REMOVED)

=> d l47 ibib abs total

L47 ANSWER 1 OF 3 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2003:37304700 BIOTECHNO
TITLE: Large carboxy-terminal parathyroid hormone (PTH) fragment with a relatively longer half-life than 1-84 PTH is secreted directly from the parathyroid gland in humans
AUTHOR: Yamashita H.; Gao P.; **Cantor T.**; Futata T.; Murakami T.; Uchino S.; Watanabe S.; Kawamoto H.; Fukagawa M.; Noguchi S.
CORPORATE SOURCE: H. Yamashita, Noguchi Thyroid Clinic, Hospital Foundation, 6-33 Noguchi-Nakamachi, Beppu, Oita 874-0932, United States.
E-mail: yama@noguchi-med.or.jp
SOURCE: European Journal of Endocrinology, (2003), 149/4 (301-306), 28 reference(s)
CODEN: EJOEEP ISSN: 0804-4643
DOCUMENT TYPE: Journal; Article
COUNTRY: United Kingdom
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2003:37304700 BIOTECHNO
AB Objective: It was discovered that an immunoreactive large carboxy-terminal parathyroid hormone (PTH) fragment (large C-PTH), likely 7-84 PTH, is present in the circulation. However, very little is known about the production and metabolism of this large C-PTH. Combining a whole molecule PTH (**whole PTH**) immunoradiometric assay (IRMA) specifically for 1-84 PTH and an intact PTH (iPTH) IRMA for the sum of 1-84 PTH and large C-PTH, we were able to assess the circulating level of this large C-PTH as well as the glandular secretion and metabolism of this large C-PTH in primary hyperparathyroidism (pHPT). Methods: This study consisted of two patient groups consisting of 77 pHPT patients with a single adenoma. Of these, 43 comprised the venous sampling study group and 70 comprised the intra-operative PTH study group. (Seven patients belonged only to the former group, 34 patients to only the latter group, and 36 patients to both groups.) Preoperatively, blood samples were drawn from the bilateral internal jugular vein by ultrasonographic guidance and from the peripheral vein (n = 43). During surgery, blood samples were drawn after anesthesia (basal level), before excision (pre-excision level) of one enlarged parathyroid gland, and at 5, 10, and 15 min post-excision (n = 70). Results: There were 26 patients whose iPTH assay levels differed by more than 10% between the right and left internal jugular. In 24 of the 26 patients, the large C-PTH levels obtained from the adenoma side were significantly higher than those from the contralateral side (117±135 vs 43±33 pg/ml, P < 0.001). The plasma **whole PTH** values decreased more rapidly than the iPTH values after parathyroidectomy (P < 0.001). Conclusions: Our study has demonstrated that the large C-PTH, likely 7-84 PTH, is directly released from the parathyroid gland in humans. Since the half-life of 1-84 PTH is much shorter than large C-PTH, likely 7-84 PTH, it would be advantageous to use an assay that specifically measures 1-84 PTH for

intra-operative monitoring of parathyroidectomy.

L47 ANSWER 2 OF 3 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2003:36826939 BIOTECHNO
TITLE: IRMA (**whole PTH**) is a more useful assay for the effect of PTH on bone that the Allergo intact PTH assay in CAPD patients with low bone turnover marker
AUTHOR: Tanno Y.; Yokoyama K.; Nakayama M.; Katoh A.; Yamamoto H.; Iwasaki Y.; **Cantor T.**; Fukagawa M.; Shigematsu T.; Hosoya T.
CORPORATE SOURCE: Dr. Y. Tanno, The Division of Kidney/Hypertension, Jikei University School of Medicine, 3-25-8 Nishi-Shinbashi, Minato-ku, Tokyo 105-8471, Japan. E-mail: tanno@jikei.ac.jp
SOURCE: Nephrology Dialysis Transplantation, (01 JUN 2003), 18/SUPPL. 3 (iii97-iii98), 6 reference(s)
CODEN: NDTREA ISSN: 0931-0509
DOCUMENT TYPE: Journal; Article
COUNTRY: United Kingdom
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2003:36826939 BIOTECHNO
AB The common intact parathyroid hormone (i-PTH) assay detects not only PTH (1-84) but also the PTH (7-84) fragment. Recently, it was reported that the PTH (7-84) fragment is an antagonist to the biological action of PTH (1-84). It was also reported that the accumulation of the PTH (7-84) fragment plays a role in skeletal resistance in haemodialysis (HD) patients. However, the role of accumulation of the PTH (7-84) fragment in continuous ambulatory peritoneal dialysis (CAPD) patients, with a different clearance rate from that of HD patients, is still unclear. Therefore, we have measured only the active form of PTH (1-84) using a new method of **whole PTH** (w-PTH) assay in 20 CAPD patients (15 male and five female; mean age 51.0±13.0 years). The mean w-PTH value was 88.5 ±14.2 pg/ml in CAPD patients, which was 42.1% of i-PTH (152.6±23.6 pg/ml). The approximate value of w-PTH was calculated using the following formula (w-PTH=0.58 x iPTH-0.4, R.sup.2=0.94). PTH (7-84) fragment was calculated by the formula i-PTH-w-PTH. The PTH (7-84) fragment/w-PTH ratio as an index of skeletal resistance, and serum alkaline phosphatase activity as an osteoblastic marker were negatively correlated (P=0.02). From these results, we concluded that the i-PTH level as calculated using the common assay method might lead to an overestimation of parathyroid function and bone turnover in CAPD patients similarly to HD patients. The w-PTH assay may be useful for more precise evaluation of PTH activity in end-stage renal disease patients.

L47 ANSWER 3 OF 3 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
DUPLICATE
ACCESSION NUMBER: 2000:30620025 BIOTECHNO
TITLE: A novel mechanism for skeletal resistance in uremia
AUTHOR: Slatopolsky E.; Finch J.; Clay P.; Martin D.; Sicard G.; Singer G.; Gao P.; **Cantor T.**; Dusso A.
CORPORATE SOURCE: Dr. E. Slatopolsky, Washington Univ. School of Medicine, Renal Division, Box 8126, 660 South Euclid Avenue, St. Louis, MO 63110, United States.
SOURCE: Kidney International, (2000), 58/2 (753-761), 31 reference(s)
CODEN: KDYIA5 ISSN: 0085-2538
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2000:30620025 BIOTECHNO

AB Background. In treating secondary hyperparathyroidism, the target level of serum intact parathyroid hormone (I-PTH) should be three to five times normal to prevent adynamic bone disease. In circulation, there is a non-(1-84) PTH-truncated fragment, likely 7-84, which, in addition to PTH 1-84, is measured by most I-PTH immunoradiometric (IRMA) assays, giving erroneously high I-PTH values. We have developed a new IRMA assay in which the labeled antibody recognizes only the first six amino acids of the PTH molecule. Thus, this new IRMA assay (**Whole PTH**) measures only the biologically active 1-84 PTH molecule. Methods. Using this new IRMA assay (**Whole PTH**) and the Nichols 'intact' PTH assay, we compared the ability of each assay to recognize human PTH (hPTH) 1-84 and hPTH 7-84 and examined the percentage of non-1-84 PTH in circulation and in parathyroid glands. Possible antagonistic effects of the 7-84 PTH fragment on the biological activity of 1-84 PTH in rats were also tested. Results. In 28 uremic patients, PTH values measured with the Nichols assay, representing a combined measurement of both hPTH 1-84 and hPTH 7-84, were 34% higher than with the Whole assay (hPTH 1-84 only); the median PTH was 523 versus 318 pg/mL ($P < 0.001$). Similar results were found in 14 renal transplant patients. In osteoblast-like cells, ROS 17.2, 1-84 PTH (10.sup.-.sup.8 mol/L) increased cAMP from 18.1 ± 1.25 to 738 ± 4.13 mmol/well. Conversely, the same concentration of 7-84 PTH had no effect. In parathyroidectomized rats fed a calcium-deficient diet, 7-84 PTH was not only biologically inactive, but had antagonistic effects on 1-84 PTH in bone. Plasma calcium was increased (0.65 mg/dL) two hours after 1-84 PTH treatment, while 7-84 PTH had no effect. When 1-84 PTH and 7-84 PTH were given simultaneously in a 1:1 molar ratio, the calcemic response to 1-84 PTH was decreased by 94%. In normal rats, the administration of 1-84 PTH increased renal fractional excretion of phosphate (11.9 to 27.7%, $P < 0.001$). However, when 1-84 PTH and 7-84 PTH were given simultaneously, the 7-84 PTH decreased the phosphaturic response by 50.2% ($P < 0.005$). Finally, in surgically excised parathyroid glands from six uremic patients, we found that 44.1% of the total intracellular PTH was the non-PTH (1-84), most likely PTH 7-84. Conclusion. In patients with chronic renal failure, the presence of high circulating levels of non-1-84 PTH fragments (most likely 7-84 PTH) detected by the 'intact' assay and the antagonistic effects of 7-84 PTH on the biological activity of 1-84 PTH explain the need of higher levels of 'intact' PTH to prevent adynamic bone disease.

```
=> file .jacob
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                        ENTRY    SESSION
FULL ESTIMATED COST                                     12.74      70.98

FILE 'CAPLUS' ENTERED AT 16:27:02 ON 03 JAN 2005
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2005 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'BIOSIS' ENTERED AT 16:27:02 ON 03 JAN 2005
Copyright (c) 2005 The Thomson Corporation.

FILE 'MEDLINE' ENTERED AT 16:27:02 ON 03 JAN 2005

FILE 'EMBASE' ENTERED AT 16:27:02 ON 03 JAN 2005
COPYRIGHT (C) 2005 Elsevier Inc. All rights reserved.

FILE 'USPATFULL' ENTERED AT 16:27:02 ON 03 JAN 2005
CA INDEXING COPYRIGHT (C) 2005 AMERICAN CHEMICAL SOCIETY (ACS)

=> cantor t/au
L48             3 FILE CAPLUS
```

```
L49          22 FILE BIOSIS
L50           9 FILE MEDLINE
L51          19 FILE EMBASE
L52           0 FILE USPATFULL

TOTAL FOR ALL FILES
L53          53 CANTOR T/AU

=> l53 and pTH and whole
L54           0 FILE CAPLUS
L55           7 FILE BIOSIS
L56           3 FILE MEDLINE
L57           9 FILE EMBASE
L58           0 FILE USPATFULL

TOTAL FOR ALL FILES
L59          19 L53 AND PTH AND WHOLE

=> dup rem
ENTER L# LIST OR (END):l59
PROCESSING COMPLETED FOR L59
L60          16 DUP REM L59 (3 DUPLICATES REMOVED)

=> l60 and antibody
L61           0 S L60
L62           0 FILE CAPLUS
L63           7 S L60
L64           0 FILE BIOSIS
L65           2 S L60
L66           1 FILE MEDLINE
L67           7 S L60
L68           0 FILE EMBASE
L69           0 S L60
L70           0 FILE USPATFULL

TOTAL FOR ALL FILES
L71           1 L60 AND ANTIBODY

=> d l71 ibib ab total

L71  ANSWER 1 OF 1     MEDLINE on STN
ACCESSION NUMBER:   2000421662     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 10916099
TITLE:              A novel mechanism for skeletal resistance in uremia.
COMMENT:            Comment in: Kidney Int. 2001 Jun;59(6):2375-6. PubMed ID:
                    11380847
AUTHOR:             Slatopolsky E; Finch J; Clay P; Martin D; Sicard G; Singer
                    G; Gao P; Cantor T; Dusso A
CORPORATE SOURCE:   Renal Division, Washington University Medical School, St.
                    Louis, MO 63110, USA.
CONTRACT NUMBER:    AM-07126 (NIADDK)
     AM-09976 (NIADDK)
     RR-00036 (NCRR)
SOURCE:             Kidney international, (2000 Aug) 58 (2) 753-61.
                    Journal code: 0323470. ISSN: 0085-2538.
PUB. COUNTRY:       United States
DOCUMENT TYPE:      (CLINICAL TRIAL)
                    Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
ENTRY MONTH:        200009
ENTRY DATE:         Entered STN: 20000915
                    Last Updated on STN: 20020911
                    Entered Medline: 20000907
```

AB BACKGROUND: In treating secondary hyperparathyroidism, the target level of serum intact parathyroid hormone (I-**PTH**) should be three to five times normal to prevent adynamic bone disease. In circulation, there is a non-(1-84) **PTH**-truncated fragment, likely 7-84, which, in addition to **PTH** 1-84, is measured by most I-**PTH** immunoradiometric (IRMA) assays, giving erroneously high I-**PTH** values. We have developed a new IRMA assay in which the labeled **antibody** recognizes only the first six amino acids of the **PTH** molecule. Thus, this new IRMA assay (**Whole PTH**) measures only the biologically active 1-84 **PTH** molecule. METHODS: Using this new IRMA assay (**Whole PTH**) and the Nichols "intact" **PTH** assay, we compared the ability of each assay to recognize human **PTH** (hPTH) 1-84 and hPTH 7-84 and examined the percentage of non-1-84 **PTH** in circulation and in parathyroid glands. Possible antagonistic effects of the 7-84 **PTH** fragment on the biological activity of 1-84 **PTH** in rats were also tested. RESULTS: In 28 uremic patients, **PTH** values measured with the Nichols assay, representing a combined measurement of both hPTH 1-84 and hPTH 7-84, were 34% higher than with the **Whole** assay (hPTH 1-84 only); the median **PTH** was 523 versus 318 pg/mL ($P < 0.001$). Similar results were found in 14 renal transplant patients. In osteoblast-like cells, ROS 17.2, 1-84 **PTH** (10-8 mol/L) increased cAMP from 18.1 +/- 1.25 to 738 +/- 4.13 mmol/well. Conversely, the same concentration of 7-84 **PTH** had no effect. In parathyroidectomized rats fed a calcium-deficient diet, 7-84 **PTH** was not only biologically inactive, but had antagonistic effects on 1-84 **PTH** in bone. Plasma calcium was increased (0.65 mg/dL) two hours after 1-84 **PTH** treatment, while 7-84 **PTH** had no effect. When 1-84 **PTH** and 7-84 **PTH** were given simultaneously in a 1:1 molar ratio, the calcemic response to 1-84 **PTH** was decreased by 94%. In normal rats, the administration of 1-84 **PTH** increased renal fractional excretion of phosphate (11.9 to 27.7%, $P < 0.001$). However, when 1-84 **PTH** and 7-84 **PTH** were given simultaneously, the 7-84 **PTH** decreased the phosphaturic response by 50.2% ($P < 0.005$). Finally, in surgically excised parathyroid glands from six uremic patients, we found that 44.1% of the total intracellular **PTH** was the non-**PTH** (1-84), most likely **PTH** 7-84. CONCLUSION: In patients with chronic renal failure, the presence of high circulating levels of non-1-84 **PTH** fragments (most likely 7-84 **PTH**) detected by the "intact" assay and the antagonistic effects of 7-84 **PTH** on the biological activity of 1-84 **PTH** explain the need of higher levels of "intact" **PTH** to prevent adynamic bone disease.

PTH on the biological activity of 1-84 **PTH** explain the need of higher levels of 'intact' **PTH** to prevent adynamic bone disease.

L19 ANSWER 6 OF 11 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
DUPLICATE
ACCESSION NUMBER: 1997:27329806 BIOTECHNO
TITLE: History of posttransfusion hepatitis
AUTHOR: Tobler L.H.; Busch M.P.
CORPORATE SOURCE: L.H. Tobler, Irwin Memorial Blood Centers, 270 Masonic Ave, San Francisco, CA 94118, United States.
E-mail: itobler@ccnet.com
SOURCE: Clinical Chemistry, (1997), 43/8 SUPPL. (1487-1493), 47 reference(s)
CODEN: CLCHAU ISSN: 0009-9147
DOCUMENT TYPE: Journal; Conference Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 1997:27329806 BIOTECHNO
AB The risk of hepatitis virus transmission from transfusions has declined dramatically from that of the 1940s when posttransfusion hepatitis (**PTH**) was first appreciated. Introduction of hepatitis B surface antigen screening and conversion to volunteer donors for **whole** -blood donations in the late 1960s and early 1970s led to substantial reduction in **PTH** cases. However, up to 10% of the recipients continued to develop **PTH**, most cases of which were attributed to an unknown non-A, non-B vital agent. Implementation of surrogate marker testing (i.e., alanine aminotransferase and anti-hepatitis B virus core antigen) for residual non-A, non-B hepatitis in the late 1980s reduced the per unit risk of **PTH** from 1 in 200 to about 1 in 400. Hepatitis C virus was discovered in 1989 and quickly was established as the causative agent of >90% of non-A, non-B **PTH**. Introduction of progressively improved **antibody** assays in the early 1990s reduced the risk of **PTH** due to hepatitis C virus to about 1 in 100 000. Although additional hepatitis viruses exist (e.g., hepatitis G virus), these appear to be minor contributors to clinical **PTH**, which has been virtually eradicated.

L19 ANSWER 7 OF 11 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
DUPLICATE
ACCESSION NUMBER: 1996:26330992 BIOTECHNO
TITLE: **PTH**-related protein is released into the mother's bloodstream during lactation: Evidence for beneficial effects on maternal calcium-phosphate metabolism
AUTHOR: Lippuner K.; Zehnder H.-J.; Casez J.-P.; Takkinen R.; Jaeger P.
CORPORATE SOURCE: Policlinic of Medicine, University Hospital,3010 Berne, Switzerland.
SOURCE: Journal of Bone and Mineral Research, (1996), 11/10 (1394-1399)
CODEN: JBMREJ ISSN: 0884-0431
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 1996:26330992 BIOTECHNO
AB Recent studies have indicated that **parathyroid hormone** -related protein (PTHrP) may have important actions in lactation, affecting the mammary gland, and also calcium metabolism in the newborn and the mother. However, there are as yet no longitudinal studies to support the notion of an endocrine role of this peptide during nursing. We studied a group of 12 nursing mothers, mean age 32 years, after they

had been nursing for an average of 7 weeks (B) and also 4 months after stopping nursing (A). It was assumed that changes occurring between A and B correspond to the effect of lactation. Blood was assayed for prolactin (PRL), PTHrP (two-site immunoradiometric assay with sheep **antibody** against PTHrP(1-40), and goat **antibody** against PTHrP(60-72), detection limit 0.3 pmol/l), intact **PTH** (iPTH), ionized calcium (Ca.sup.2.sup.+), 25- hydroxyvitamin D.sub.3 (25(OH)D.sub.3) and 1,25-dihydroxyvitamin D.sub.3 (1,25(OH).sub.2D.sub.3), alkaline phosphatase (alkP), as well as for creatinine (Cr), protein, phosphorus (P), and total calcium (Ca). Fasting 2-h urine samples were analyzed for Ca excretion (CaE) and renal phosphate threshold (TmP/GFR). PRL was significantly higher during lactation than after weaning (39 ± 10 vs. 13 ± 9 μg/l; p = 0.018) and so was PTHrP (2.8 ± 0.35 vs. 0.52 ± 0.04 pmol/l; p = 0.002), values during lactation being above the normal limit (1.3 pmol/l) in all 12 mothers. There was a significant correlation between PRL and PTHrP during lactation (r = 0.8, p = 0.002). **Whole** blood Ca.sup.2.sup.+ did not significantly change from A (1.20 ± 0.02 mmol/l) to B (1.22 ± 0.02, mmol/l), whereas total Ca corrected for protein (2.18 ± 0.02 mmol/l) or uncorrected (2.18 ± 0.02 mmol/l) significantly rose during lactation (2.31 ± 0.02 mmol/l, p = 0.003 and 2.37 ± 0.03 mmol/l, p = 0.002, respectively). Conversely, iPTH decreased during lactation (3.47 ± 0.38 vs. 2.11 ± 0.35 pmol/l, A vs. B, p = 0.02). Serum-levels of 25(OH)D.sub.3 and 1,25(OH).sub.2D.sub.3 did not significantly change from A to B (23 ± 2.3 vs. 24 ± 1.9 ng/ml and 29.5 ± 6.0 vs. 21.9 ± 1.8 pg/ml, respectively). Both TmP/GFR and P were higher during lactation than after weaning (1.15 ± 0.03 vs. 0.86 ± 0.05 mmol/l GF, p = 0.003 and 1.25 ± 0.03 vs. 0.96 ± 0.05 mmol/l, p = 0.002, respectively) as was alkP (74.0 ± 7.1 vs. 52.6 ± 6.9 U/l, p = 0.003). CaE did not differ between A and B (0.015 ± 0.003 vs. 0.017 ± 0.003 mmol/l GF, A vs. B, NS). We conclude that lactation is accompanied by an increase in serum PRL. This is associated with a release of PTHrP into the maternal blood circulation. A rise in total plasma Ca ensues, probably in part by increased bone turnover as suggested by the elevation of alkP. **PTH** secretion falls, with a subsequent rise of TmP/GFR and plasma P despite high plasma levels of PTHrP.

L19 ANSWER 8 OF 11 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
ACCESSION NUMBER: 1991:21314326 BIOTECHNO
TITLE: Incidence of Anti-Hepatitis C virus **antibodies** in Non-A, Non-B Post-Transfusion hepatitis in an area of Northern Italy
AUTHOR: Elia G.F.; Magnani G.; Belli L.; Formentini A.; Iacono S.; Marchelli Pincolini S.S.; Lecchini R.; Bernuzzi G.; Fiaccadori F.
CORPORATE SOURCE: Universita' di Parma, Servizio Anestesia, Ospedsle Maggiore di Parma, Via A Gramsci 14,I-43100 Parma, Italy.
SOURCE: Infection, (1991), 19/5 (336-339)
CODEN: IFTNAL ISSN: 0300-8126
DOCUMENT TYPE: Journal; Article
COUNTRY: Germany, Federal Republic of
LANGUAGE: English
SUMMARY LANGUAGE: German; English
AN 1991:21314326 BIOTECHNO
AB A total of 210 patients consecutively submitted to heart surgery at the Parma University Hospital and transfused with 1,898 units of blood were followed after transfusion in order to evaluate both the incidence of anti-hepatitis C virus (HCV) seroconversion in non-A, non-B post-transfusion hepatitis (**PTH**-NANB) cases and the usefulness of the screening for anti-HCV in comparison with that for serum glutamic pyruvic transaminase (SGPT) values in preventing **PTH**-NANB transmission. Fifteen recipients developed **PTH**-NANB (group A);

ten of them (66.6%) showed anti-HCV seroconversion within 3-12 months. Eight of the ten anti-HCV positive patients developed chronic hepatitis, but none of the five **PTH**-NANB anti-HCV negative did. None of the 15 controls (group B) randomly chosen among the patient population showed anti-HCV seroconversion. A close correlation with the transmission of **PTH** was showed by anti-HCV positivity but not by SGPT elevation in blood donors. Eleven of 172 blood products transfused to group A but none of 139 products transfused to group B were anti-HCV positive. The incidence of elevated SGPT values was similar between the two groups of the transfused blood products. Nevertheless, the correlation observed between anti-HCV positivity and SGPT levels in the blood products involved in **PTH** confirms the need to exclude blood donors with abnormal SGPT values. On the **whole**, anti-HCV screening of donors showed a predictive value higher than that of SGPT (100% vs. 53.3%), allowing a minor blood donation exclusion. The percentage of anti-HCV seroconversion observed in **PTH**-NANB is probably underestimated because of the limits of the ELISA method we used for the detection of anti-HCV.

L19 ANSWER 9 OF 11 LIFESCI COPYRIGHT 2005 CSA on STN
ACCESSION NUMBER: 85:76395 LIFESCI
TITLE: Localization of **parathyroid hormone** -like substance in squamous cell carinomas: An immunoperoxidase study with ultrastructural correlation.
AUTHOR: Ilardi, C.F.; Faro, J.C.
CORPORATE SOURCE: Dep. Lab., Long Island Jewish Med. Cent., New Hyde Park, NY 11042, USA
SOURCE: ARCH. PATHOL. LAB. MED., (1985) vol. 109, no. 8, pp. 752-755.
DOCUMENT TYPE: Journal
FILE SEGMENT: T
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Histologic sections of squamous cell carcinomas of four hypercalcemic patients were investigated for the presence of **parathyroid hormone** (**PTH**)-like substance. The Sternberger-peroxidase-antiperoxidase-immunoperoxidase technique utilizing monospecific **antibody** to **whole** (1 to 84) bovine **PTH** demonstrated immunoreactive material in all cases. Electron microscopy of the four tumors revealed dense-core secretory granules resembling those seen in parathyroid chief-cell adenomas. The hypercalcemia associated with some nonmetastatic squamous cell carcinomas is associated with the production of **PTH**-like substance.

L19 ANSWER 10 OF 11 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
ACCESSION NUMBER: 1982:13254064 BIOTECHNO
TITLE: **Parathyroid hormone** assay
AUTHOR: Chih Kao P.
CORPORATE SOURCE: Dep. Lab. Med., Mayo Clin., Rochester, MN, United States.
SOURCE: Mayo Clinic Proceedings, (1982), 57/9 (596-597) CODEN: MACPAJ
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
AN 1982:13254064 BIOTECHNO
AB Antiserum GP-235 recognizes intact **whole**-molecule **PTH** and C-terminal fragments of 44-68 and 53-84 amino acid sequences but does not recognize the N-terminal 1-34 amino acid sequence. A radioimmunoassay was developed with this antiserum, and 118 normal subjects of both sexes from 20 to 55 years of age were studied. The serum **PTH** concentrations in all of these subjects were less than 70 μleq/ml, which is our upper limit of normal. The radioimmunoassay developed with this antiserum can differentiate normal subjects from patients with

primary hyperparathyroidism, hypoparathyroidism, chronic renal failure, or hypercalcemia caused by malignancy.

L19 ANSWER 11 OF 11 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
ACCESSION NUMBER: 1981:12196086 BIOTECHNO
TITLE: Functional receptors for epidermal growth factor on human osteosarcoma cells
AUTHOR: Shupnik M.A.; Tashjian Jr. A.H.
CORPORATE SOURCE: Lab. Toxicol., Harvard Sch. Pub. Hlth, Harvard Med. Sch., Boston, MA 02115, United States.
SOURCE: Journal of Cellular Physiology, (1981), 109/3 (403-410)
CODEN: JCLLAX
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
AN 1981:12196086 BIOTECHNO
AB Previous studies have shown that epidermal growth factor (EGF) stimulates bone resorption in organ culture via a prostaglandin-mediated pathway, and that there are specific receptors for EGF on mouse bone (Tashjian and Levine, '78; Shupnik et al., '80). The present study demonstrates that a clonal line of human osteosarcoma cells, G-292 (Clone A141B1) has specific, high-affinity receptors for EGF and responds to treatment with EGF by increased prostaglandin production. Binding of .sup.1.sup.2.sup.5I-EGF to G-292 cells exhibited a prolonged plateau phase (6 hours); thereafter, binding slowly declined (t.half. = 6 hours) to 30-40% of the maximal level. This decrease in cell-associated .sup.1.sup.2.sup.5I-EGF was prevented by leupeptin (50 μg/ml). EGF binding was of high affinity (K(d) = 1.9 X 10.sup.-.sup.9 M) and was to a single class of non-interacting binding sites. Pretreatment of cells for 48 hours with EGF caused a maximum threefold increase in PGE.sub.2 production, with a half-maximal response at 9.8 X 10.sup.-.sup.1.sup.0 M EGF. Increased PGE.sub.2 production was detectable within 2 hours and the constant presence of EGF was needed to maintain the response. Although EGF is mitogenic in several other systems, it did not increase DNA synthesis in the osteosarcoma cells. EGF treatment also did not increase medium or intracellular cyclic AMP in these cells, although **parathyroid hormone** and exogenous PGE.sub.2 (200 ng/ml) increased cyclic AMP three- to tenfold over control levels. Pretreatment with EGF decreased the level of subsequent .sup.1.sup.2.sup.5I-EGF binding; receptor number decreased to 30-40% of control after 48 hours of treatment, and the half-maximal effect occurred with pretreatment concentrations of 1.6 X 10.sup.-.sup.9 M EGF. In all respects tested, the binding and biological actions of EGF on the human osteosarcoma cells were the same as those in **whole** mouse bone. G-292 cells thus provide a convenient model system to study EGF action on osseous tissue.